TECHNICAL NOTE
NO. TN96-2

BODY FLUID BALANCE DURING EXERCISE-HEAT EXPOSURE

by

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March 1996

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REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. AGENCY USE ONLY (Leave blank)	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	March 1996	Technical Note

4. TITLE AND SUBTITLE

Body Fluid Balance During Exercise-Heat Stress

5. FUNDING NUMBERS

6. AUTHOR(S)

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7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)

US Army Research Institute of Environmental Medicine
Kansas Street
Natick, MA 01760-5007

8. PERFORMING ORGANIZATION REPORT NUMBER

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)

US Army Medical Research and Materiel Command
Fort Detrick
Frederick, MD 21702-5012

10. SPONSORING/MONITORING AGENCY REPORT NUMBER

11. SUPPLEMENTARY NOTES

12a. DISTRIBUTION/AVAILABILITY STATEMENT

Approved for public release; distribution is unlimited.

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

During exercise, sweat output often exceeds water intake; producing a body water deficit or hypohydration. The water deficit affects both intracellular and extracellular volume. It also results in plasma hypertonicity and hypovolemia. Muscular strength and endurance can be reduced by hypohydration, but the effects are not clear-cut. Aerobic exercise tasks are likely to be adversely affected by hypohydration; with the potential being greater in warm environments. Hypohydration increases heat storage and reduces one's ability to tolerate heat strain. The increased heat storage is mediated by reduced sweating rate and reduced skin blood flow for a given core temperature. Hyperhydration has been suggested to reduce thermal strain during exercise in the heat, however, data supporting that notion are not robust.

14. SUBJECT TERMS	15. NUMBER OF PAGES
Hypohydration, Dehydration, Hyperhydration, Heat Stress, Exercise Performance	38
	16. PRICE CODE

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
Unclassified	Unclassified	Unclassified	

EXECUTIVE SUMMARY

During exercise, sweat output often exceeds water intake; producing a body water deficit or hypohydration. The water deficit affects both intracellular and extracellular volume. It also results in plasma hypertonicity and hypovolemia. Muscular strength and endurance can be reduced by hypohydration, but the effects are not clear-cut. Aerobic exercise tasks are likely to be adversely affected by hypohydration; with the potential being greater in warm environments. Hypohydration increases heat storage and reduces one's ability to tolerate heat strain. The increased heat storage is mediated by reduced sweating rate and reduced skin blood flow for a given core temperature. Hyperhydration has been suggested to reduce thermal strain during exercise in the heat, however, data supporting that notion are not robust.

INTRODUCTION

Persons performing exercise in the heat will often incur a body water deficit. Generally, the person dehydrates during exercise because of fluid nonavailability or a mismatch between thirst and body water requirements. In these instances, the person starts to exercise as euhydrated but incurs an exercise-heat mediated dehydration over a prolonged period. This scenario is common for many athletic and occupational settings; however, in the military, particularly during combat, the person might start to exercise while already hypohydrated (Draper & Lombardi, 1986). There are also several sports (e.g., boxing, power lifting, wrestling) where athletes will purposely achieve hypohydration prior to competition (Zambraski, et al., 1976). These athletes want to compete in a lower weight class to gain a size advantage over competitors.

If a dehydrated person exercises in the heat, they will incur significant adverse effects. Dehydration will increase physiologic strain, decrease exercise performance and negate the thermoregulatory advantages conferred by high aerobic fitness (Buskirk et al., 1958; Cadarette et al., 1984) and heat acclimation (Buskirk et al., 1958; Sawka et al., 1983). In addition, devastating medical consequences can occur if dehydrated persons perform strenuous exercise in the heat. For example, Massachusetts State Police recruits were limited access to water during training sessions in the summer of 1988, and 11 of 50 class members were hospitalized with serious heat injuries; 2 underwent kidney dialysis, and one required a liver transplant and later died (Commonwealth of Massachusetts, 1988). Another example, is that dehydration induced heat stroke is believed responsible for 20,000 deaths among Egyptian troops during the 1967 six-day war with Israel (Hubbard et al., 1982).

This chapter will review human hydration status and temperature regulation during exercise in the heat. Throughout this chapter "euhydration" will refer to normal body water content; "hypohydration" refers to body water deficit; and "dehydration" refers to the loss of body water.

BODY WATER LOSS

Physical exercise routinely increases total body metabolism by 5-15 times resting levels to support skeletal muscle contraction. Approximately 70% to 90% of this energy is released as heat and needs to be dissipated to achieve body heat balance. Depending on the climatic conditions the relative contributions of evaporative and dry (radiative and conductive) heat exchange to the total heat loss will vary. The hotter the climate the greater the dependence on evaporative heat loss, and thus on sweating. Therefore, a substantial volume of body water may be lost via sweating to enable evaporative cooling in hot climates (Sawka & Pandolf, 1990).

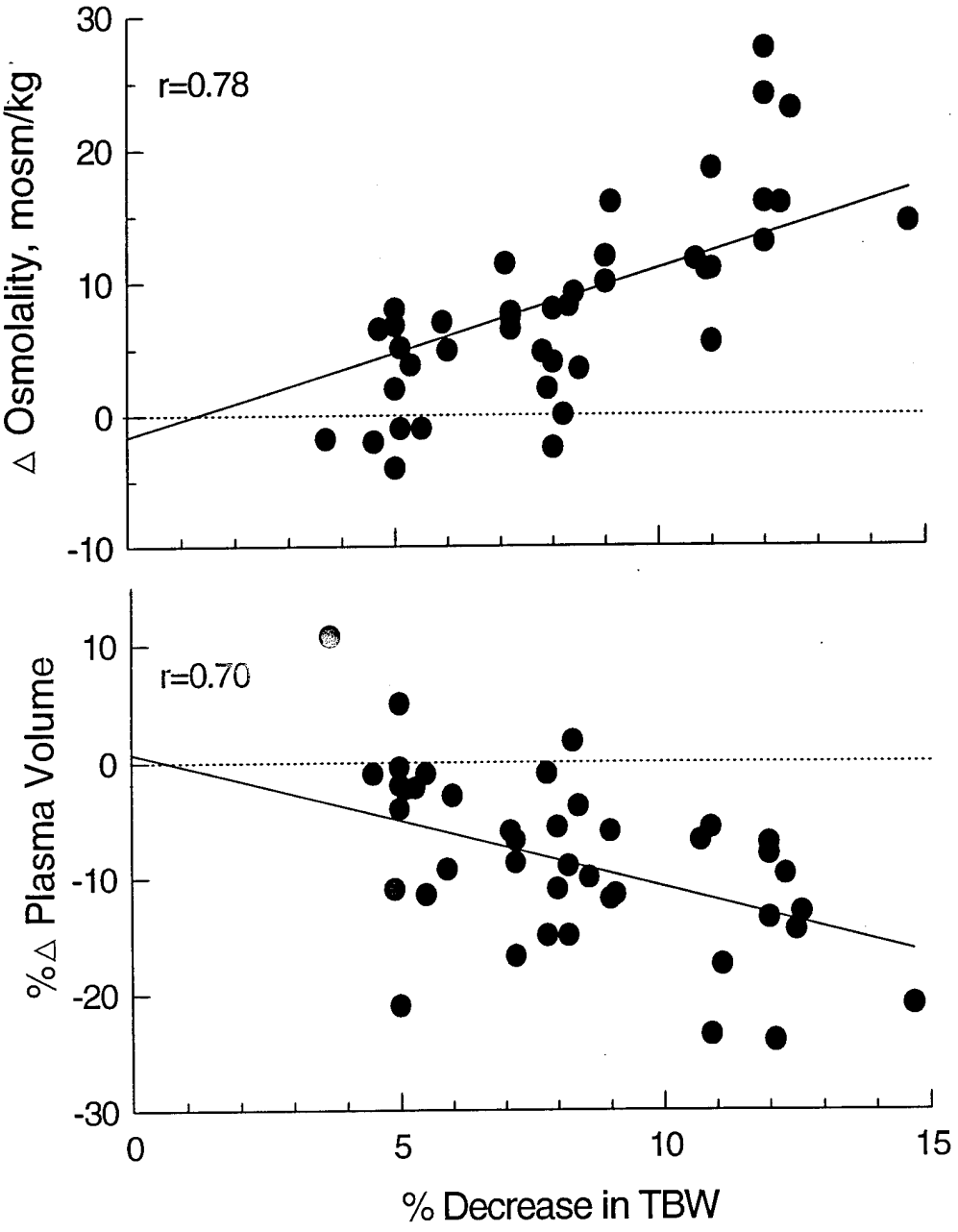
A person's sweating rate is dependent upon the climatic conditions, clothing worn and exercise intensity. Soldiers in the desert often have sweating rates of 0.3 to 1.2 $L \cdot h^{-1}$ while performing military activities (Adolph & assoc., 1947; Molnar et al., 1946). Persons wearing protective clothing often have sweating rates of 1 to 2 $L \cdot h^{-1}$ while performing light intensity exercise (Levine et al., 1990; Speckman et al.,, 1988). Likewise, athletes performing high intensity exercise commonly have sweating rates of 1 to 2.5 $L \cdot h^{-1}$ while in the heat (Sawka and Pandolf, 1990). During these situations, a principal problem is to avoid dehydration by matching fluid consumption to sweat loss. Since thirst provides a poor index of body water needs, persons will dehydrate by 2% to 8% of their body weight during situations of prolonged high sweat loss (Greenleaf, 1992).

Water is the largest component of the human body, comprising 45 to 70 percent of body weight. The average male (75 kg) is composed of about 45 L of water, which corresponds to about 60 percent of body weight. Since adipose tissue is about 10 percent water and muscle tissue is about 75 percent water, a person's total body water depends upon their body composition (Sawka and Pandolf, 1990). In addition, muscle water and glycogen content parallel each other probably because of the osmotic pressure exerted by glycogen granules within the muscle's sarcoplasm (Neufer et al., 1991). As a result, trained athletes have a relatively greater total body water, than their sedentary counterparts, by virtue of a smaller percentage body fat and a higher skeletal muscle glycogen concentration.

The water contained in body tissues is distributed between the intracellular and extracellular fluid spaces. Dehydration mediated by sweating will influence each fluid space as a consequence of free fluid exchange. Nose and colleagues (1983) determined the distribution of body water loss among the fluid spaces as well as among different body organs. They thermally dehydrated rats by 10% of body weight, and after the animals regained their normal core temperature, the body water measurements were obtained. The water deficit was apportioned between the intracellular (41%), and extracellular (59%) spaces; and among the organs, 40% from muscle, 30% from skin, 14% from viscera and 14% from bone. Neither the brain nor liver lost significant water content. They concluded that hypohydration results in water redistribution largely from the intra- and extracellular spaces of muscle and skin in order to maintain blood volume.

Figure 1 presents resting plasma volume and osmolality values for heat acclimated persons when hypohydrated at various levels of body water loss. Sweat induced dehydration will decrease plasma volume and increase plasma osmotic pressure in proportion to the level of fluid loss. Plasma volume decreases because it provides the precursor fluid for sweat, and osmolality increases because sweat is ordinarily hypotonic relative to plasma (Kubica et al., 1983; Senay, 1968). Sodium and chloride are primarily responsible for the elevated plasma osmolality (Senay, 1968). It is the plasma hyperosmolality which mobilizes fluid from the intracellular to the extracellular space to enable plasma volume defense in hypohydrated subjects.

Some athletes use diuretics to reduce their body weight. Diuretics increase the rate of urine formation and generally result in the loss of solutes (Weiner and Mudge, 1985). Commonly used diuretics include: thiazide (e.g. Diuril), carbonic anhydrase inhibitors (e.g. Diamox) and furosemide (e.g. Lasix). Diuretic-induced hypohydration generally results in an iso-osmotic hypovolemia, with a much greater ratio of plasma loss to body water loss than either exercise or heat induced hypohydration (Hubbard & Armstrong, 1988). Relatively less intracellular fluid is lost after diuretic administration since there a not an extracellular solute excess to stimulate redistribution of body water.


30
20
10
0
-10
r=0.78
△ Osmolality, mosm/kg
10
0
-10
-20
-30
r=0.70
%△ Plasma Volume
0
5
10
15
% Decrease in TBW

EXERCISE PERFORMANCE & TOLERANCE

MUSCULAR STRENGTH AND ENDURANCE

Muscular strength has been examined in 12 studies, of which four demonstrated a strength reduction after dehydration (see Table 1). Of the four studies that demonstrated strength reductions, hypohydration was achieved by fluid restriction in three (Bosco & Terjung, 1968; Bosco et al., 1974; Houston et al., 1981) and by a combination of exercise and heat exposure in the fourth (Webster et al., 1988). Therefore, prolonged fluid restriction, perhaps accompanied by a caloric deficit (Bosco & Terjung, 1968; Houston et al., 1981), was the dehydration method that most often reduced muscular strength. The magnitude of water deficit appeared to influence the frequency with which muscular strength reductions were reported. Only one (Bosco & Terjung, 1968) of the five studies, employing less than a 5% reduction in body weight, reported a strength reduction; three of the seven studies employing between a 5% to 8% reduction in body weight reported strength reductions. Interestingly, the magnitude of water deficit did not appear to influence the magnitude of strength reduction, as decreases approximating 10% were reported for each study. Finally, it appears that the upper body muscles are more likely than lower body muscles to show a strength reduction from hypohydration (Bosco and Terjung, 1968; Webster et al., 1988).

Muscular endurance was evaluated in four studies, of which two (Bijlani & Sharma, 1980; Torranin et al., 1979) demonstrated a reduction after hypohydration. The two studies that found no change in muscular endurance did not report their dehydration procedure (Mnatzakanian & Vaccaro, 1982; Serfass et al., 1984); but the other two studies used either thermal (Torranin et al., 1979) or exercise and thermal (Bijlani & Sharma, 1980) dehydration procedures. There were no systematic differences among these four studies in the magnitude of water deficit, muscle group tested, or test methodology.

Torranin et al., (1979) evaluated the isometric endurance of a small muscle group (hand grip) and the isotonic endurance of large muscle groups (arm and leg).

Table 1. Influence of hypohydration on muscular strength and endurance.

Study	Dehydration Procedure	ΔWT	Method	Strength	Endurance/ Power
Ahlman & Karvonen [2]	Sauna & Exercise	-2kg	Back leg lifts	NC	–
Greenleaf et al. [30]	Exercise in heat	-3%	Isometric	NC	–
Bosco et al. [9]	Fluid restriction	-3%	Isometric	↓(11%)	–
Bijlani & Sharma [6]	Exercise in heat	-3%	Isometric	NC	↓
Greenleaf et al. [31]	Fluid restriction	-4%	Isometric	NC	–
Saltin [65]	Sauna, Exercise in heat, exercise	-4%	Isometric Arm Cranking	NC –	– ↓(13 to 37%)
Torranin et al. [86]	Sauna	-4%	Isometric Isotonic	– –	↓(31%) ↓(29%)
Mnatzakanian & Vaccaro [48]	?	-4%	Isokinetic	NC	NC
Tuttle [87]	Exercise & heat	-5%	Isometric	NC	–
Jacobs [39]	Heat	-5%	Wingate Test	–	NC
Serfass et al. [78]	?	-5%	Isometric	NC	NC
Webster et al. [89]	Exercise in heat, sauna	-5%	Wingate Test	NC in leg strength ↓(7%) in shoulder & chest strength	↓(21%) peak power ↓(10%) average power
Bosco et al. [8]	Fluid restriction	-6%	Isometric Isotonic	↓(3%) –	– ↓
Singer & Weiss [80]	?	-7%	Isometric	NC	–
Houston et al. [36]	Fluid restriction	-8%	Isokinetic Supramax. run	↓(11%) –	– NC

NC= No change from euhydration.

Despite the use of very diverse methodology, they found a consistent 30% reduction in muscular endurance when hypohydrated. They speculated that during the hypohydration experiments, a greater muscle temperature might have mediated the reduced muscular endurance. The muscular endurance experiments were conducted approximately 1 h after the subjects had finished dehydrating in an 80°C sauna.

Bijlani and Sharma (1980) performed their muscular endurance experiments either immediately after or within 30 min of dehydrating their subjects in a 41°C environment. In addition, it appeared that all of their muscular testing (when eu- and hypohydrated) was conducted in a warm (30-35°) environment. They present a figure demonstrating an inverse relationship between the control (euhydration) muscular endurance values and the dry bulb temperature. Therefore, an elevated muscle temperature could have mediated the reduced muscular endurance during the hypohydration experiments (Edwards & Lippold, 1972; Petrofsky & Lind, 1975).

Anaerobic exercise performance was evaluated in four studies, of which two employed Wingate type tests (Jacobs, 1980; Webster et al., 1988), and two studies employed supramaximal endurance tests (Houston et al., 1981; Nielsen et al., 1981). Jacobs (1980) performed a comprehensive evaluation of anaerobic exercise performance (Wingate test) in subjects when they were euhydrated and when they were hypohydrated by 2%, 4% and 5% of their body weight. This investigator found that hypohydration did not alter anaerobic exercise performance or post-exercise blood lactate values. On the other hand, Webster and colleagues (1988) reported that hypohydration (5% body weight) resulted in a 21% reduction in peak power and a 10% reduction in average power during a 40 sec Wingate test. Both of these studies used similar methodologies so that their disparate results are not easily explained.

Nielsen et al., (1981) had subjects perform a supramaximal (105% $\dot{V}O_2max$) cycle ergometer test both when euhydrated and when hypohydrated (3% of body weight). Exercise performance was decreased when subjects were hypohydrated with diuretics (-18%), sauna (-35%) and previous exercise (-44%). These reductions in supramaximal exercise performance were related to an elevation of plasma potassium concentration as well as an increase in skeletal muscle temperature.

Houston et al., (1981) reported that hypohydration (8% of body weight) did not affect supramaximal (~1 min) treadmill run performance. In this experiment, dehydration was achieved by fluid and food restriction over several days, and the subjects were not exposed to heat stress.

AEROBIC EXERCISE

Table 2 presents a summary of studies concerning the influence of hypohydration on maximal aerobic power and physical work capacity. In a temperate environment, a body water deficit of less than 3% body weight did not alter maximal aerobic power. Maximal aerobic power was decreased (Buskirk et al., 1958; Caldwell et al., 1984; Webster et al., 1988) in three of the five studies when hypohydration equaled or exceeded 3% body weight. Therefore, a critical water deficit (3% body weight) might exist before hypohydration reduces maximal aerobic power in a temperate environment. In a hot environment, Craig and Cummings (1966) demonstrated that small (2% body weight) to moderate (4% body weight) water deficits resulted in large reduction of maximal aerobic power. Likewise, their data indicate a disproportionately larger decrease in maximal aerobic power with an increased magnitude of body water deficit. It seems that environmental heat stress has a potentiating effect on the reduction of maximal aerobic power elicited by hypohydration.

The physical work capacity for progressive intensity aerobic exercise was decreased when hypohydrated. Physical work capacity was decreased by marginal (1% - 2% body weight) water deficits that did not alter maximal aerobic power (Armstrong et al., 1985; Caldwell et al., 1984) and the reduction was larger with increasing water deficit. Clearly, hypohydration resulted in much larger decrements of physical work capacity in hot as compared to temperate environments (Craig and Cummings, 1966). It appears that the thermoregulatory system, perhaps via increased body temperatures, has an important role in the reduced exercise performance mediated by a body water deficit.

TABLE 2. Effect of hypohydration on maximal aerobic power and physical work capacity.

Study	Dehydration Procedure	WT Loss	Climate	Exercise Mode	Maximal Aerobic Power	Physical Work Capacity
Armstrong et al. [4]	Diuretics	1%	Neutral	TM	NC	↓ (6.2%)
Caldwell, et al. [13]	Exercise, diuretics, sauna	2%	Neutral	CY	NC	↓ (7 W)
		3%	Neutral	CY	↓ (8%)	↓ (21 W)
		4%	Neutral	CY	↓ (4%)	↓ (23 W)
Pichan et al. [62]	Fluid restriction	1%	Hot	CY	–	↓ (6%)
		2%	Hot	CY	–	↓ (8%)
		3%	Hot	CY	–	↓ (20%)
Neilsen et al. [58]	Diuretic	-3%		CY	–	↓ (18%)
	Sauna	-3%	Neutral	CY	–	↓ (35%)
	Exercise	-3%		CY	–	↓ (44%)
Saltin [65]	Sauna, heat, exercise, diuretics	4%	Neutral	CY	NC	↓ (?)
Burge et al. [10]	Exercise & fluid restriction	3%	Neutral	Rowing	NC	↓ (5%)
Craig & Cummings [18]	Heat	2%	Hot	TM	↓ (10%)	↓ (22%)
		4%	Hot	TM	↓ (27%)	↓ (48%)
Buskirk et al. [11]	Exercise, heat	5%	Neutral	TM	↓ (0.22 L/min)	–
Webster et al. [89]	Exercise, eat, sauna	5%	Neutral	TM	↓ (7%)	↓ (12%)
Houston et al. [36]	Fluid restriction	8%	Neutral	TM	NC	–

NC= No change from euhydration. TM= treadmill exercise. CY= cycling exercise.

A reduced maximal cardiac output might be the physiological mechanism by which hypohydration decreases an individual's maximal aerobic power and physical work capacity. Hypohydration is associated with a decreased blood (plasma) volume during both rest and exercise. A decreased blood volume increases blood viscosity and can reduce venous return. During maximal exercise, viscosity mediated increased resistance and a reduced cardiac filling could both decrease stroke volume and cardiac output. Several investigators (Allen et al., 1977; Sproles et al., 1976; Saltin, 1964b) have reported a tendency for reduced cardiac output when hypohydrated during short-term moderate intensity exercise.

It is not surprising that environmental heat stress potentiates the hypohydration mediated reduction in maximal aerobic power. For euhydrated individuals, environmental heat stress alone decreases maximal aerobic power by ~7% (Sawka et al., 1985a). In the heat, the superficial skin veins reflexively dilate to increase cutaneous blood flow and volume. The redirection of blood flow to the cutaneous vasculature could decrease maximal aerobic power by: (a) reducing the portion of cardiac output perfusing contracting muscles or, (b) decreasing the effective central blood volume, central venous pressure and thus reduce venous return and cardiac output. If a person was hypohydrated and encountered environmental heat stress, they would be hypovolemic and still have to simultaneously perfuse the cutaneous vasculature and contracting skeletal muscles. A substantial volume of blood can be redirected to the skin; and therefore removed from the effective central circulation and not available to perfuse the skeletal muscles (Rowell, 1986). As a result, both environmental heat stress and hypohydration can act independently to limit cardiac output and therefore oxygen delivery during maximal exercise.

Studies have also demonstrated that moderate levels of hypohydration can impair endurance exercise performance. Armstrong and colleagues (1985) studied the effects of a body water deficit on competitive distance running performance. They had athletes compete in 1,500, 5,000 and 10,000 meter races when euhydrated and when hypohydrated. Hypohydration was achieved by diuretic administration (furosemide) which decreased body weight by 2% and plasma volume by 11%. Running performance was impaired at all race distances, but to a greater extent in the longer races (~5% for the 5,000 and 10,000 m) than the shorter race (3% for

1,500 m). Burge et al. (1993) recently examined whether hypohydration (3% body weight loss) affected simulated 2,000 m rowing performance. They found that on average, it took 22 seconds longer to complete the task when hypohydrated compared to when euhydrated. Average power was reduced 5% by hypohydration.

Surprisingly few investigators have documented the effects of dehydration on human tolerance to submaximal exercise in the heat. Adolph and associates (1947) performed experiments in the California deserts during 1942 and 1943. In those experiments, subjects attempted endurance (2 to 23 hours) walks (at 4 to 6.5 $km \cdot h^{-1}$ h-1) in the desert (T_a ~ 38°C) and either were allowed to drink water ad libitum or refrained from drinking. They reported that one of fifty-nine (2%) and eleven of seventy soldiers (16%) suffered exhaustion from heat strain during a desert walk when they did or did not drink, respectively. In subsequent experiments, they reported that one of fifty-nine subjects (2%) and fifteen of seventy subjects (21%) suffered exhaustion from heat strain during an attempted eight-hour desert walk when they did and did not drink, respectively. The subjects' magnitude of dehydration was not provided in either set of experiments. Ladell (1955) had subjects attempt a 140-minute walk in a hot (T_a = 38°C) environment while ingesting different combinations of salt and water. They reported that exhaustion from heat strain occurred in 9 of 12 (75%) experiments when receiving neither water or salt, and 3 of 41 (7%) experiments when receiving only water. Sawka and colleagues (1985) had subjects attempt treadmill walks (~25% $\dot{V}O_2max$ for 140 min) in a hot-dry (T_a = 49° C, rh=20%) environment when euhydrated and when hypohydrated by 3%, 5%, and 7% of their body weight. All eight subjects completed the euhydration and 3% hypohydration experiments, and seven subjects completed the 5% hypohydration experiments. For the 7% hypohydration experiments, six subjects discontinued after completing only (mean) 64 minutes. Clearly, dehydration increases the incidence of exhaustion from heat strain.

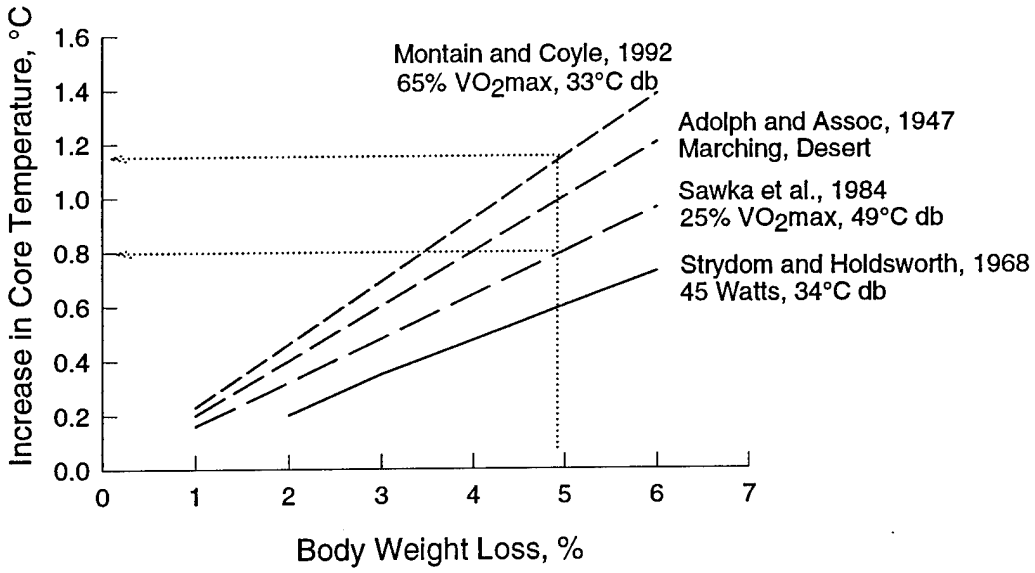
To address whether hypohydration alters heat tolerance, Sawka and colleagues (1992) had subjects walk to voluntary exhaustion when either euhydrated or hypohydrated (8% of total body water). The experiments were designed so that the combined environment (T_a = 49° C, rh = 20%) and exercise intensity (47% $\dot{V}O_2max$) would not allow thermal equilibrium and heat exhaustion would eventually

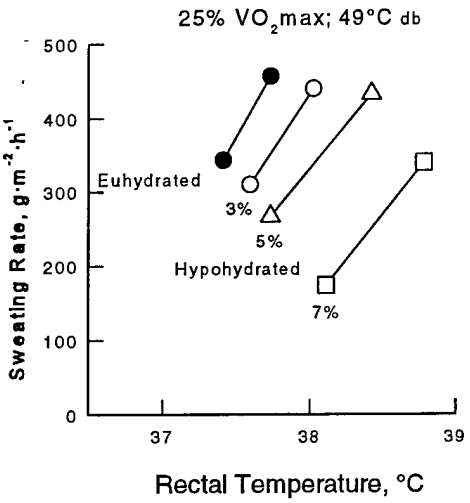
occur. Hypohydration reduced tolerance time (121 to 55 min), but more importantly, hypohydration reduced the core temperature that a person could tolerate. Heat exhaustion occurred at a core temperature ~0.4 °C lower when hypohydrated than when euhydrated. These findings suggest that hypohydration not only impairs exercise performance, but also reduces tolerance to heat strain.

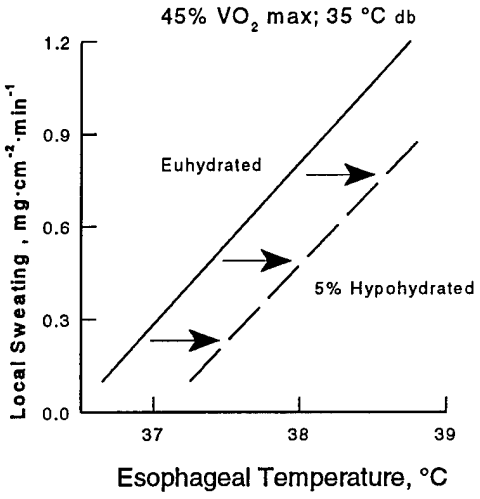
TEMPERATURE REGULATION

Hypohydration increases core temperature responses during exercise in temperate (Cadarette et al., 1984; Grande et al., 1959) and hot (Sawka et al, 1985) climates. A critical deficit of 1% of body weight elevates core temperature during exercise (Ekblom et al., 1972). As the magnitude of water deficit increases, there is a concomitant graded elevation of core temperature during exercise heat stress (Montain and Coyle, 1992; Sawka et al., 1985). Figure 2 illustrates relationships between body water loss and core temperature elevations reported by studies which examined several water deficit levels. The magnitude of core temperature elevation ranges from 0.1 to 0.23°C for every percent body weight lost (Adolph & assoc., 1947; Sawka et al., 1985; Strydom and Holdsworth, 1968; Montain and Coyle, 1992). These studies suggest that the core temperature elevation, for a given water deficit, becomes greater with increased exercise intensity. In addition, hypohydration negates the thermoregulatory advantages conferred by high aerobic fitness and heat acclimation during exercise in the heat (Buskirk et al., 1958; Cadarette et al., 1984, Sawka et al., 1983).

Hypohydration is reported to be associated with both reduced (Moroff and Bass, 1965; Senay, 1968; Strydom and Holdsworth, 1968) or unchanged (Claremont et al, 1976; Strydom et al., 1966; Swamy et al., 1981) sweating rates during exercise at a given metabolic rate. However, investigators reporting no change in sweating rate usually observed an elevated core temperature. Figure 3 (left) shows with increased hypohydration levels a systematic reduction in total body sweating rate for a given core temperature during exercise in the heat (Sawka et al., 1985). Likewise, Figure 3 (right) presents the local sweating response to hypohydration (5% body weight) during exercise in the heat (Sawka et al., 1989). This figure suggests that hypohydration


Increase in Core Temperature, °C
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0.0
0
1
2
3
4
5
6
7
Body Weight Loss, %
Montain and Coyle, 1992
65% VO2max, 33°C db
Adolph and Assoc, 1947
Marching, Desert
Sawka et al., 1984
25% VO2max, 49°C db
Strydom and Holdsworth, 1968
45 Watts, 34°C db


25% VO2max; 49°C db
Sweating Rate, g·m-2·h-1
500
400
300
200
100
0
Euhydrated
3%
5%
Hypohydrated
7%
37
38
39
Rectal Temperature, °C


45% VO2 max; 35 °C db
Local Sweating , mg·cm-2·min-1
1.2
0.9
0.6
0.3
0.0
Euhydrated
5% Hypohydrated
37
38
39
Esophageal Temperature, °C

increases the threshold temperature for the onset of sweating, but does not alter the sensitivity (slope) of the sweating response to increases in body temperature.

The physiological mechanisms mediating the altered control of sweating when hypohydrated are not clearly defined. Both the singular and combined effects of plasma hyperosmolality and hypovolemia have been suggested as mediating the reduced sweating response during exercise-heat stress (Sawka, 1992). Plasma osmolality changes may relate to tonicity changes in the extracellular fluid bathing the hypothalamic neurons (Kozlowski et al., 1980; Senay, 1979). Silva and Boulant (1984) have demonstrated that in rat brain slices, there are preoptic-anterior hypothalamic neurons which are both thermosensitive and osmosensitive. Such data suggests a central interaction between thermoregulation and body water regulation (Hori et al., 1988).

Isotonic hypohydration alone can impair heat loss and increase core temperature during exercise-heat exposure (Claremont et al., 1976; Nadel et al., 1980; Fortney et al., 1981a). Studies (Fortney et al., 1981b; Nadel et al., 1980) have demonstrated reported that isotonic hypohydration reduces skin blood flow for a given core temperature, and therefore the potential for dry heat exchange. Fortney et al. (1981) have provided a rationale as to why an iso-osmotic hypohydration might reduce skin blood flow and sweating rate. They theorized that hypovolemia might reduce cardiac pre-load and alter the activity of atrial baroreceptors which have afferent input to the hypothalamus. Therefore, a reduced atrial filling pressure might modify neural information to the hypothalamic thermoregulatory centers which control skin blood flow and sweating. Subsequent studies (Mack et al., 1988; Gaddis & Elizondo, 1984) have demonstrated that acute unloading of atrial baroreceptors during exercise with periods of lower body negative pressure impairs heat loss and increases core temperature.

HYPERHYDRATION

Hyperhydration, or greater than normal body water, has been suggested to improve, above euhydration levels, thermoregulation and exercise-heat performance.

The concept that hyperhydration might be beneficial for exercise performance arose from the adverse consequences of hypohydration. It was theorized that body water expansion might reduce the cardiovascular and thermal strain of exercise by expanding blood volume and reducing blood tonicity; thereby improving exercise performance.

Studies that have directly expanded blood volume (e.g. infusion) have usually reported decreased cardiovascular strain (Hopper et al., 1988; Nose et al., 1990; Sawka et al., 1983) during exercise, but disparate results on heat dissipation (Deschampes et al., 1989; Nose et al., 1990; Sawka et al., 1983) and exercise-heat performance (Deschampes et al., 1989; Luetkemeier & Thomas, 1994). Studies that have attenuated plasma hyperosmolality during exercise-heat stress generally report improved heat dissipation (Fortney et al., 1984; Harrison et al., 1978; Nielsen, 1974; Sawka et al., 1989), but have not addressed exercise performance.

Table 3 provides a chronological review of studies evaluating hyperhydration effects on thermoregulation. Generally, most studies reported lower core temperatures during exercise after hyperhydration. Three of the seven studies (Moroff & Bass, 1965; Nielsen, 1974; Lyons et al., 1990) reported higher sweating rates with hyperhydration. In all cases, heart rate was lower during exercise with hyperhydration. Together, these findings support the notion that hyperhydration can reduce the thermal and cardiovascular strain of exercise.

The mechanism(s) responsible for the lower exercise core temperatures when hyperhydrated remain unclear. Overdrinking before exercise, in several studies (Moroff & Bass, 1965; Nielsen, 1971) lowered body core temperature prior to exercise. This was likely due to the caloric cost of warming the ingested fluid. Exercise *per se* did not exacerbate the difference that existed prior to exercise. Hyperhydration in these studies, therefore, apparently did not improve heat dissipation during the exercise period. Other studies (Lyons et al., 1990; Moroff and Bass, 1965; Nielsen 1974), however, reported greater exercise sweating rates when hyperhydrated. Grucza et al. (1987) found that sweating was initiated earlier when hyperhydrated.

Table 3. Hyperhydration effects on thermoregulation.

Study	Year	Pre-Exercise Hydration Treatments	Exercise-Environmental Conditions	Temperature		Sweat Rate
				Core	Skin	
Moroff & Bass [52]	1965	2 L water vs no water	90 min treadmill (Ta=49°C)	↓(0.3°C)	–	↑
Nielsen et al. [57]	1971	1.5 L water vs no water or 1.0 L 2% saline	60 min cycle (~50%$\dot{V}O_2$max) (Ta=20°C)	↓(0.5°C) ↑(0.3°C)	– –	– –
Greenleaf & Castle [30]	1971	2.5-3.0 L water vs ad libitum	70 min cycle (~50%$\dot{V}O_2$max) (Ta=24°C)	↓(0.25°C, NS)	NC	↑(NS)
Nielsen [56]	1974	1.5 L water vs no water vs 1.5 L 2-3% saline	60 min cycle (45%$\dot{V}O_2$max) (Ta=30°C)	↓ ↑	– –	↑ ↓
Gisolfi & Copping [28]	1974	1 L water vs no water vs 1 L with rehydration	120 min treadmill (75%$\dot{V}O_2$max) (Ta=33°C)	↓(0.2°C) ↓(0.8°C)	– –	NC NC
Nadel et al. [53]	1980	2.0 L water with ADH vs euhydration	30 min cycle (55%$\dot{V}O_2$max) (Ta=35°C)	↓(0.12°C, NS)	–	–
Grucza et al. [32]	1987	2.0 L water vs euhydration	45 min cycle (~52%$\dot{V}O_2$max) (Ta=23°C)	↓(0.4°C) ↓(△0.2°C)	NC	↓
Candas et al.[14]	1988	0.5 L isotonic solution vs euhydration	4h intermittent cycle (70 W) (Ta=36°C)	NC	NC	NC
Lyons et al.[46]	1990	1.5 L water vs 1.5 L water with glycerol or ad libitum	90 min treadmill (~60%$\dot{V}O_2$max) (Ta=42°C)	↓(0.7°C) NC	– –	↑ NC

NC= No difference from euhydration. NS= Not statistically different from euhydration. Ta=ambient temperature.

The findings of these latter studies suggest that hyperhydration may improve heat dissipation during exercise-heat stress.

The thermal benefits of hyperhydration do not appear dependant on acclimation state. Moroff and Bass (1965) examined the effect of hyperhydration on the heat acclimation response. Repeated hyperhydration prior to exercise during 9 days of exercise-heat stress did not affect the acclimation response. Furthermore, overdrinking produced similar effects on core temperature, heart rate and sweating regardless whether hyperhydration was induced before or after the heat acclimation protocol.

While many studies have attempted to induce hyperhydration by overdrinking water or water-electrolyte solutions, these approaches have produced only transient expansion of body water. One problem often encountered is that much of the fluid overload is rapidly excreted. Recently, evidence has accrued that greater fluid retention can be achieved with an aqueous solution containing glycerol. Riedesel et al. (1987) reported that following hyperhydration with a glycerol solution compared to water alone, subjects excreted significantly less of the water load. Improved fluid retention after hyperhydration with glycerol solutions has been confirmed by others (Freund et al., in review; Lyons et al., 1990) and there are reports that hyperhydration can be maintained for prolonged periods with repeated glycerol intake (Koeningsberg et al., 1991).

Lyons et al. (1990) examined whether glycerol-mediated hyperhydration improved thermoregulatory responses to exercise-heat stress. Subjects completed three trials in which they exercised in a hot (42°C) climate. For one trial, fluid ingestion was restricted to 5.4 ml/kg body weight, and in the other two trials subjects ingested water (21.4 ml/kg) with or without a bolus of glycerol (1 g/kg). Ninety minutes after this hyperhydration period subjects began exercise. Glycerol ingestion increased fluid retention 30% compared to drinking water alone. During exercise, glycerol hyperhydration produced a higher sweating rate and substantially lower core temperatures (0.7°C) compared to control conditions and water hyperhydration.

Few studies have examined whether hyperhydration improves exercise performance or heat tolerance. Blyth and Burt (1961) were the first to report the effects of hyperhydration on performance during exercise-heat stress. Their subjects ran to exhaustion in a hot climate (49°C) when normally hydrated and when hyperhydrated by drinking 2 L of fluid 30 min prior to exercise. When hyperhydrated, thirteen of 18 subjects ran longer before exhaustion. The average time to exhaustion (17.3 vs 16.9 min) d5d not, however, reach statistical significance. More recently, Luetkemeier and Thomas (1994) examined whether hypervolemia improved cycling performance. They reported that blood volume expansion (+450-500 ml) increased simulated time trial performance 10% (81 vs 90 min). No study to date has examined whether hyperhydration improves exercise-heat tolerance.

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